LETTERS TO THE EDITOR

Acute upside down reversal of vision in vertebrobasilar ischaemia

Acute upside down reversal of vision is an uncommon and little known phenomenon consisting of transient complete 180 degree inversion of the visual image. The pathogenesis and the anatomical sites of this dysfunction are unknown. Lesions involving cortical areas, mainly the parieto-occipital region, or the vestibulocerebellar system have occasionally been documented.[1-5]

We observed two patients who experienced this bizarre visual illusion, both revealing features of vertebrobasilar ischaemia.

Patient 1, a 69 year old woman, was admitted because two weeks earlier she had experienced sudden malaise, sweating, nausea, vomiting, right occipital headache, followed by a 180 degree vertical inversion of the visual image, lasting about 20 minutes. Two similar episodes had occurred the day before admission. On admission the patient was alert, cooperative and well-oriented. The neurological examination was normal. In particular, no neurophthalmological abnormalities were found on clinical examination. Blood parameters, urine, chest radiograph and ECG proved normal. Cervical radiographs revealed osteoarthritis with osteophytes and narrowing of disc space C6-C7. EEG was normal. Brainstem auditory evoked potentials (BAER) revealed increased latency of V wave on right stimulation. Cerebral CT and MRI (figure) showed an ischaemic-like lesion, 2 cm diameter, in the right cerebellar hemisphere in the territory of the medial branch of the posterior inferior cerebellar artery (PICA), without mass effect. Moderate periventricular white matter abnormalities coexisted. Four vessel cerebral angiography revealed a right vertebral artery stenosis (50%) and two small arteriovenous malformations on the course of the right ascending cervical artery; a decreased flow in the basilar artery was noted. Ticlopidine 250mg daily was given and the patient was discharged. No further attacks or other neurological disturbances occurred during the next two years.

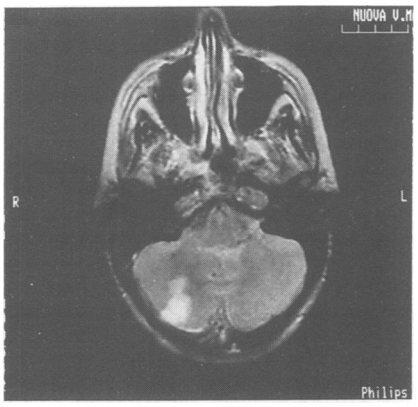

Figure Axial MR T2-weighted image showing a high signal area in the territory of the medial branch of the right PICA.

Case 2, a 52 year old woman, with a 40 year history of bilateral chronic otitis with residual deafness, had recent recurrent episodes of sudden sweating, nausea, occipital headache, dizziness, sometimes followed by a transient loss of consciousness. The whole episode usually lasted about 30–40 minutes. Frequently, at the height of dizziness, the patient experienced a 180 degree vertical visual inversion of images. These episodes occurred monthly. On admission, the neurological examination was normal. Rare, isolated, left-sided jerks of horizontal nystagmus were revealed by ENG. A 20 mmHg difference between right and left brachial arterial pressure (right > left) was noted. Ultrasound vascular investigations (Doppler cortico-vertebral echotomography and cerebral transcranial Doppler) revealed a left subclavian artery stenosis with a steal syndrome. Cerebral SPECT, CT and MRI proved normal. BAER was unavailable due to the peripheral deficit caused by chronic otitis. Cerebral angiography was refused. Flunarizine 10mg daily was given and the patient was discharged with a warning to avoid strenuous physical activities, especially those involving upper limbs and neck. No further episodes were reported in the subsequent six months.

These two women presented episodes of vertically inverted vision—upside down phenomenon—associated with clinical signs and symptoms of vertebrobasilar insufficiency. Both reported transient visual inversion of 180 degrees, which was bilateral, of sudden onset and lacking subjective impression of movement (rotatory or torsional). In the first patient, neuroimaging revealed a right hemispheric cerebellar infarction. In the second, a vertebrobasilar failure due to a left subclavian stenosis was detected. The pathogenetic mechanism underlying upside down visual inversion is unknown. Since the visual images enter the retina inverted, it may be assumed that the upside down phenomenon results from a transient failure of the mechanisms mediating reinversion, even though the anatomical structures involved are unknown. In earlier observations,[2] parietal and/or occipital lesions were sometimes found, suggesting a cortical origin of the dysfunction, probably affecting the integrative control of spatial vision. More recent cases,[1 3-5] documented with neuroimaging techniques, revealed an association with vestibular/cerebellar lesions, that is, vertebrobasilar TIAs, Wallenberg's syndrome and also cerebellar infarct in two cases.[1 5] In our patients, the relationship between the upside down visual inversion and the signs and symptoms of vertebrobasilar insufficiency, without evidence of cerebral damage, supports the idea that a transient inactivation of infratentorial structures may cause this unusual phenomenon. Besides the integrity of the visual system, space visual perception needs a flow of extraretinal information, mediated by the vestibular and cerebellar systems.[6 7] It has been suggested that damage to such structures may cause tilt and complete inversion of the visual space.[6] The upside down phenomenon may occur following dysfunctions at various levels of the complex vestibulocerebellar-ocular system mediating the stabilisation of the visual function so that cortical involvement is not indispensable.

ANDREA STRACCIARI
MARIA GUARINO
GABRIELE CIUCCI*
PAOLO PAZZAGLIA
Neurology Unit, S Orsola-Malpighi Hospital, Division of Neurology, Bologna
S. Maria delle Croci Hospital, Ravenna, Italy

Correspondence to: Dr Stracciari, Servizio di Neurologia, Policlinico S Orsola-Malpighi, via Albertoni 15, 40138 Bologna, Italy

1 Ropper AH. Illusion of tilting of the visual environment. Report of five cases. *J Clin Neurophthalmol* 1983;3:147–51.
2 Solms M, Kaplan-Solms K, Saling M, Miller P. Inverted vision after frontal lobe disease. *Cortex* 1988;24:499–509.
3 Steiner I, Shahin R, Melamed E. Acute "upside down" reversal of vision in transient vertebrobasilar ischemia. *Neurology* 1987;37: 1685–6.
4 Mehler MF. Complete visual inversion in vertebrobasilar ischaemic disease. *J Neurol Neurosurg Psychiatry* 1988;51:1236–7.
5 Charles N, Froment C, Rode G, *et al*. Vertigo and upside down vision due to an infarct in the territory of the medial branch of the posterior inferior cerebellar artery caused by dissection of a vertebral artery. *J Neurol Neurosurg Psychiatry* 1992;55:188–189.
6 Hornsten G. Constant error of visual egocentric orientation in patients with acute vestibular disorder. *Brain* 1979;102: 685–700.
7 Gloning K, Hoff H. Cerebral localisation of disorders of higher nervous activity. Clinical, anatomical and physiological aspects. In: PJ Vinken and GW Bruyn, eds, *Handbook of clinical neurology, Vol 3*, Amsterdam: North Holland, 1969, 22–47.

Subcortical environmental reduplication: SPECT findings in a patient with a right thalamocapsular haemorrhage

Recently Nighoghossian *et al*[1] reported the case of a patient with a previous history of a left fronto-basal haemorrhage, who developed environmental reduplication following an infarction of the retrolenticular portion of the right internal capsulae. SPECT revealed right fronto-parietal cortical hypoperfusion. A similar delusional misidentification syndrome was described previously in a patient with a right thalamic haemorrhage, but its functional correlate using SPECT was not studied.[2] We describe the neuroimaging and cognitive functioning of a case of environmental reduplication associated with a right thalamocapsular haemorrhage.

A 71 year old ambidextrous man suddenly developed a left-sided weakness and mild dysarthria. He had had hypertension but no history of previous cerebrovascular events. Neurological examination revealed a dense left hemiplegia, and a left sensory loss affecting all modalities. Visual fields were full, and there was no evidence of visual or auditory extinction on double simultaneous stimulation. He showed left hemispatial neglect on drawing, and on a letter cancellation task he only crossed targets on the right side of the paper. He did not deny his left hemiplegia, but he had a tendency to attribute it to previous "chest problems". He reported a feeling of nonbelonging of his paralysed left arm, and also said that he had three left legs and a strange left arm crossed over his chest. The patient said that he could walk almost normally and repeatedly tried to walk unaided despite his dense hemiplegia. He was alert and oriented to time and person, but not to place. While he